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Abstract

This paper presents an overlapping generations model where pollution, private and public healths are all determinants of longevity. Public expenditure, financed through labour taxation, provide both public health and abatement. We study the complementarity between the three components of longevity on welfare and economic stability. At the steady state, we show that an appropriate fiscal policy may enhance welfare. However, when pollution is heavily harmful for longevity, the economy might experience aggregate instability or endogenous cycles. Nonetheless, a fiscal policy, which raises the share of public spending devoted to health, may display stabilizing virtues and rule out cycles. This allows us to recommend the design of the public policy that may comply with the dynamic and welfare objectives.

JEL classification: J10; O40; Q56; C62.

Keywords: Longevity; Pollution; Welfare; Complex dynamics.

1 Introduction

Data on life expectancy and the causes of death are often seen as a relevant basis for assessing agents' health status and adjusting public policies. Since the mid-twentieth century, the health status of individuals has greatly improved in

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the world and thus, life expectancy has considerably grown[1]. These longevity gains are mainly due to substantial progress that has been made in reducing mortality due to infectious diseases, improving sources of drinking-water and basic sanitation while significant advances has been achieved in medicine and access to healthcare (WHO (2014)). The economic literature has been very prolific with regards to the contribution of health, in particular through longevity, in the development process (Mirowsky and Ross (1998), Blackburn and Cipriani (2002), Chakraborty (2004), Cervelatti and Sunde (2005), Chakraborty and Das (2005)). In richer economies, individuals may afford medical care and medicines, they are often more educated and thus adopt healthier lifestyles meanwhile public authorities are capable of funding more public health services and developing access to public health systems. Healthier agents are more productive and invest more, growth is promoted and, in turn, the enhanced development drives improvements in health status.

This virtuous cycle might however be put at risk as the development process has came along with negative externalities like pollution and environmental deterioration. In fact, regarding recent striking and acute episodes of urban air pollution, the World Health Organization (WHO (2009)) has listed among the leading global risks for health and mortality, pollution and environmental deterioration (outdoor air pollution, indoor air pollution, unsafe water etc). Say otherwise, worldwide health risks are in transition: While population is globally ageing, owing to successes against infectious diseases, people face nowadays new burdens of chronic and acute diseases, and environmental factors are a big part of these "modern" risks. Even more, in 2014 during the WHO's annual assembly that held in Geneva, new estimates were released to confirm that air pollution is now the world's largest single environmental health risk. According to WHO, in 2012, at a global scale, around 7 millions people died as a result of air exposure. In addition, 8% of lung cancer deaths should be attributable to deteriorated environmental conditions. Similarly, unsafe water might be responsible for 88% of diarrhoeal deaths. Then, it becomes vital to clearly evaluate and understand the role of these health risks to prevent from a possible economic contraction or to fight against their negative effects on longevity and welfare.

While both pollution and health status became key determinants of households longevity, they of course display opposite effects. In addition, they differ along, at least, one dimension. Individuals have a negligible influence on global pollution, especially at a macroeconomic level where pollutants can be seen as non-local ones. Moreover, global costs of abating pollution or preserving environmental conditions are often disproportionate with regards to individuals capacity to afford them. Then, it seems that those actions are mainly undertaken by public authorities. As for health, the argument is more disputable. Even if governments remain main contributors to world health spending, individuals can invest in their own health capital. For instance, such efforts include annual health screening, adoption of healthy life styles, but also out-of-pocket

[1]Even though some disparities remain among genders or/and developed and developing countries, we can observe both a convergence in life expectancy and a reduction in the cross-country dispersion of longevity (see Becker *et al.* (2003)).

expenses for essential medication and private health services. Finally, we argue that all these three components are complement inputs in the production of health status as private health efforts may induce larger improvements in life expectancy when they come along with public efforts to maintain a good environmental quality and to provide efficient public health care systems.

The goal of the paper is to show that the aforementioned interactions deeply affect the dynamic behavior of the economy. In particular, we assess whether the noxious impact of pollution on longevity might challenge the well-known two-way causality established between health and economic development in the long term. We show that these complement variables (private and public health and pollution) might indeed lead to irreversibility to the extent that they might prevent one economy to experience a stable and sustainable growth path. Formally, we emphasize the destabilising role of pollution on the dynamic behavior of the economy and underscore the occurrence of structural instability as well as endogenous cycles. These results call for a deeper exploration of what should be an appropriate public policy in order to maintain the development process or/and to restore the stability property of the dynamic behavior of the economy. As evoked previously, two types of actions can be undertaken to sustain a good health status: On the one hand, public authorities may reduce harmful pollution flows; On the other hand, they could provide health protection services or improve preventive health programmes. We argue that a well-designed public policy may exhibit both stabilizing virtues and improve the long-run welfare of economic agents.

To do so, we consider an overlapping generations model with production and endogenous longevity. Each young agent works for a competitive wage and survives to the start of the old age. Nevertheless, she is alive only for a fraction of the second period of life and only cares about their second period consumption while alive. In order to extend her time length, she may incur investment in her own health, by comparing the marginal benefit of investing in health with its opportunity cost, that is a reduction of savings. In addition, her life expectancy is positively influenced by public health expenditure while it is negatively impacted by the deterioration of the environment. Through labour income taxation, the government does not only provide health expenditure, but also pollution abatement. The latter expenses reduce the pollution stock that would otherwise raise with aggregate production. No matter the level of the tax rate, the fiscal policy consists in setting the sharing rule of public funds, that is the share devoted to public health (and respectively abatement).

In this framework, there exists a unique steady state. Taking the sharing rule as a policy parameter, we argue that it displays two opposite effects on longevity, and therefore, on capital accumulation and welfare. On the one hand, increasing the amount of public health care raises longevity. On the other hand, it generates a harmful feedback effect as abatement is cut down. We show that when pollution weakly affects longevity and/or the share of public expenditure devoted to public health is low, the first effect dominates, whereas when pollution becomes a crucial determinant of longevity and/or the public policy more favorable towards public health, the second effect dominates. The

optimal policy design in a second best framework is even precisely featured in the case of a Cobb-Douglas production function.

The complementarity between health and pollution has also strong implications on the dynamic path of physical capital accumulation and stability properties of the economy. Indeed, when the pollution effect on longevity is negligible, the steady state is stable, with monotonic convergence. However, it looses its stability when pollution becomes heavily damaging. Then, the sustainability of the development process is reconsidered as the economy experiences aggregate instability. This shift in the stability property occurs through the occurrence of endogenous cycles (Hopf bifurcation). These fluctuations are explained by the increasing weight of pollution and the complementary it induces. Indeed, a growing sequence of physical capital implies more public health expenses, but also more pollution. When pollution is significantly detrimental to longevity, the latter may decrease, pushing down the saving rate. Therefore, capital accumulation slows down, entailing a reversal of the dynamic path.

This asks for evaluating the role played by the public policy. In fact, a policy designed by a larger support towards public health might be stabilizing, by reducing the range of parameters for instability. However, before making any policy recommendation, one should also take care of the outcome of such a policy on the stationary welfare. Indeed, from our previous analysis, we have stated that such a policy might be welfare damaging when the initial support to public health is already large. In contrast, this is no longer the case when initially public funds are mainly devoted to abatement. In this last case, the two objectives of stabilisation and welfare maximisation may be reconciled.

Following Chakraborty (2004) who introduces a survival probability to old age that depends on public health expenditure only, several papers have endogenised mortality in overlapping generations growth models. For instance, Bhattacharya and Qiao (2007) consider that both private and public expenditure define the longevity, while in Jouvet *et al.* (2010), longevity raises with private health investment, but suffers from pollution. Finally, several recent papers rather consider that the survival probability depends on public health and pollution (see for instance Pautrel (2008, 2009), Varvarigos (2010), Palivos and Varvarigos (2014) and Raffin and Seegmuller (2014)). We make a bridge between these different specifications assuming that longevity depends on pollution, private and public health. However, Palivos and Varvarigos (2014) is surely the contribution closest related to ours. Considering a framework with learning-by-doing productive externalities, they show that the economy may asymptotically converge to the long run growth rate only if pollution abatement provided by the government is substantial. Otherwise, the economy experiences endogenous cycles. Our results and methodology are for all that quite different. In contrast to them, we show that reducing the public effort of abatement can stabilize the economy and we rather highlight the destabilizing role of pollution on the development pattern of the economy. In their framework, the pollution externality is not *per se* the source of endogenous fluctuations.

The paper is organized as follows. In Section 2, we present the model. In Section 3, we define an intertemporal equilibrium and show the existence and

uniqueness of the steady state. We analyse the effects of the public policy on the steady state in Section 4. In Section 5, we explore the dynamic properties of our framework. The stabilizing role of the policy is discussed in Section 6 and finally, Section 7 concludes. Many technical details are relegated to Appendices.

2 The model

2.1 Households

We consider an overlapping generations model, where time is discrete, $t = 0, 1, ..., \infty$ and the population size of a young generation is constant and normalized to unity. When young, the representative agent lives during the whole period. However, she may survive to old age with a probability $\pi_t \in [0, 1)$. When old, agents consume an amount c_{t+1} at each moment of time. Since the focus of our attention is on the links between pollution and life expectancy, we voluntary choose to abstract from consumption choices in the first period of time. Consequently, preferences of an individual born at date t are defined over the second period consumption and are represented by the following utility function:

$$U_t = \pi_t u(c_{t+1}) \tag{1}$$

In our framework, longevity is endogenous but even crucially depends on a health indicator denoted by θ_t. For tractability reasons, let us consider the following explicit function for life expectancy that satisfies usual properties (see for instance Blackburn and Cipriani (2002), Cervelatti and Sunde (2003), Chakraborty (2004), Castelló-Climent and Doménech (2008) or Palivos and Varvarigos (2014)):

$$\pi_t \equiv \pi(\theta_t) = \frac{b\theta_t}{1 + \theta_t}, \tag{2}$$

with $0 < b < 1$. Therefore, $\pi(\theta_t)$ is increasing from $\pi(0) = 0$ to $\pi(+\infty) = b$ and is strictly concave with θ_t.

When young, agents are endowed with one unit of labour, which they supply inelastically to firms. They may also influence their life expectancy by investing in their own health. Typically, these expenditure (x_t) encompass all out-of-pocket expenses for basic medication or clinical services provided by private practitioners or the cost of buying nutrients, organic food and the like. Alternatively, they may use their labour income (w_t) for savings (s_t) in order to ensure consumption streams when old[2] . Moreover, a tax $\tau \in [0, 1)$ is levied on their revenue in order to finance any kinds of public expenditure. Thus, the first period budget constraint is written as:

$$x_t + s_t = (1 - \tau)w_t \tag{3}$$

[2]Notice that in our set-up, we only focus on the link between pollution and mortality rather than morbidity. The deterioration of health status and the induced loss of productivity has already been explored in papers like Williams (2002, 2003) or Raffin (2012).

During their second period of life, agents retire and they only consume their remunerated savings[3]:

$$R_{t+1}s_t = c_{t+1}\pi_t, \tag{4}$$

where R_{t+1} denotes the interest rate under complete capital depreciation. Notice that, following Chakraborty (2004), we assume a perfect annuity market, meaning that the return of total savings of young agents is distributed among all survival old households.[4]

In addition and beyond the sole private health spending, we argue that longevity may also be shaped by publicly funded health services (denoted by η_t), which obviously enhance the health status, while the latter decline through the harmful effects of the pollution stock, P_t.[5] Accordingly, let us express the health indicator as:

$$\theta_t = \frac{x_t^{\alpha}\eta_t^{1-\alpha}}{P_t^{\beta}} \tag{5}$$

with $0 \leqslant \alpha \leqslant 1$ and $\beta \geqslant 0$. As in Bhattacharya and Qiao (2007), this equation captures the possible interactions between public and private components in the health sector, *i.e.* their complementarity. In particular, public health care programs such as provision of clinical facilities, hospitals, sanitation or disease-control measures increase the productivity of private health investments rather than replace them. The parameter α accounts for the share of private health investments in total health expenditure: The higher α, the more sensitive is the health indicator to private investments relative to public ones. But in contrast to Bhattacharya and Qiao (2007), we introduce an additional element which is the pollution stock that comes to reduce the efficiency of both types of health spending, as soon as $\beta > 0$.

Finally, in order to perform a clear and relevant analysis, we consider the following explicit instantaneous CRRA utility function:

$$u(c) = \frac{c^{1-\sigma}}{1-\sigma} \tag{6}$$

with $0 < \sigma < 1$ the degree of concavity of $u(c)$, which ensures that utility is increasing with life expectancy. Pollution and public health care being given, individuals maximize the utility function (1) substituting (2), (5) and (6), facing the two budget constraints (3) and (4), and $x_t \geqslant 0$. The first order condition (FOC) yields:

$$\frac{1}{1+\theta_t}\frac{\alpha}{x_t} \leq \frac{1-\sigma}{\sigma s_t} \tag{7}$$

[3]So far we do not consider that agents may influence their longevity when old, through curative treatment for instance. We only consider life expectancy at birth, so that longevity depends on the living conditions experienced during young age.

[4]Following this hypothesis, π_t can alternatively be interpreted as longevity or life expectancy.

[5]Notice that in our set-up, we only focus on the link between pollution and mortality rather than morbidity. The deterioration of health status and the induced loss of productivity has already been explored in papers like Williams (2002, 2003) or Raffin (2012).

with equality if $x_t > 0$.[6] This equation reflects the trade-off agents face to when choosing the amount of private health spending. On the one hand, they invest in their own health in order to live longer and to increase their utility; On the other hand, these expenses reduce the revenue to be saved for future consumption and thus lower future utility. Consequently, economic growth and longevity compete to the extent that investing in private health diverts away resources from good production, despite the fact that it extends the length of time. A deeper analysis of equation (7) allows us to state that there is no equilibrium without private health expenditure[7]:

Lemma 1 *Agents always invest a strictly positive amount of their income in private health expenditure, so that* $x_t > 0 \ \forall t \geqslant 0$.

Proof. See Appendix A. ■

Equation (7) that holds with an equality captures the arbitrage between investing in capital (s_t) and private health (x_t). On one side, because we have assumed a perfect annuity market, the marginal return of capital, $R_{t+1}u'(c_{t+1})$, is independent of agents' health status, θ_t. On the other side, the marginal return of private health, $(\partial\pi_t/\partial x_t)\sigma u(c_{t+1})$, positively depends on the induced marginal longevity gain. Due to the concavity property of π_t, the return on health investment is thus lower when the health status is already high. In that case, agents are less likely to spend in private health and this triggers more savings.

Obviously, a direct effect of a larger value of the parameter α is a reallocation of resources towards health, which is detrimental to physical capital accumulation.

2.2 Public sector

As mentioned previously, public authorities intervene in the economy through the provision of two types of public services: First, the government provides public health expenditure (η_t); Second, she may also engage in environmental protection actions (G_t) in order to reduce harmful effects of pollution on health. We consider that due to the extensively large costs associated with pollution abatement, only the government may afford them. Since both expenditure are

[6]Let us note that the second order condition (SOC) is also satisfied. Indeed, the FOC can be rewritten:

$$\frac{\partial\pi_t}{\partial x_t}\sigma u(c_{t+1}) - R_{t+1}u'(c_{t+1}) \leqslant 0$$

We deduce that the SOC is given by:

$$\frac{\partial^2\pi_t}{\partial x_t^2}\sigma u(c_{t+1}) - \left(\frac{R_{t+1}}{\pi_t} + \frac{c_{t+1}}{\pi_t}\frac{\partial\pi_t}{\partial x_t}\right)\left[\frac{\partial\pi_t}{\partial x_t}\sigma u'(c_{t+1}) - R_{t+1}u''(c_{t+1})\right] < 0$$

[7]Note that Lemma 1 holds even if we consider a CES function over private and public health. Therefore, even though public and private health expenses are not necessary to have a positive longevity, agents always invest in private health.

financed thanks to labour income taxation, they can be expressed as:

$$\eta_t = \mu\tau w_t \quad (8)$$
$$G_t = (1-\mu)\tau w_t \quad (9)$$

where $\mu \in (0,1]$ accounts for the time invariant design of the public policy (or sharing rule), that is the share of public revenue devoted to public health spending[8]. Hence, any increase in μ reduces just as much the effort of public abatement.

2.3 Pollution

In our set-up, what matters as a key determinant of life expectancy is the stock of pollution, rather than the flow of polluting emissions. Hence, we need to describe the dynamic evolution of this variable to account for the deterioration of the environmental quality over time. As standard in the literature (see for instance John and Pecchenino (1994), Withagen (1995), Jouvet *et al.* (2005)), the pollution stock evolves according to the following law of motion:

$$P_{t+1} = (1-m)P_t + \epsilon_1 Y_t - \epsilon_2 G_t \quad (10)$$

with $m \in (0,1)$, $\epsilon_1 \geqslant 0$, $\epsilon_2 \geqslant 0$, and $P_0 > 0$ given. In other words, environmental quality is damaged by streams of pollution induced by the production process but may be enhanced thanks to public spending. As for the parameter m, it captures ecological inertia phenomena, since nature can not entirely absorb pollution flows at each period of time. Finally, parameters ϵ_1 and ϵ_2 reflect the dirtiness degree of production and the efficiency of public abatement, respectively.

2.4 Production

There is a unique final good that is produced by a continuum of unit size of competitive firms using the neo-classical technology $Y_t = F(K_t, L_t)$, where Y_t is aggregate production, K_t aggregate capital and L_t aggregate labour. The production function displays usual properties and is homogeneous of degree 1. Let $k_t \equiv K_t/L_t$ denote the capital per young household and $f(k_t) \equiv F(k_t, 1)$ the intensive form of the production function. In order to lead a proper analysis of the equilibrium and the dynamics of the economy, we assume the following:

Assumption 1 *$f(k)$ is a continuous function defined on $[0,+\infty)$ and C^2 on $(0,+\infty)$, strictly increasing ($f'(k) > 0$) and strictly concave ($f''(k) < 0$). There exists $\widetilde{k} > 0$ such that $f(\widetilde{k})/\widetilde{k} - f'(\widetilde{k}) > \left(1 + \alpha\frac{\sigma}{1-\sigma}\right)/(1-\tau)$. Defining $s(k) \equiv f'(k)k/f(k) \in (0,1)$ as the capital share in total income and $\rho(k) \equiv [f'(k)\,k/f(k) - 1]\,f'(k)\,/\,[kf''(k)] > 0$ as the elasticity of capital-labour substitution, we further assume $\rho(k) > \max\{2s(k); 1-s(k)\}$ and $s(k) < 1/2$.*

[8] Note that we assume $\mu \neq 0$ to exclude a zero utility coming from a nil longevity.

Profit maximization yields:

$$w_t = f(k_t) - k_t f'(k_t) \equiv w(k_t) \quad (11)$$
$$R_t = f'(k_t) \equiv R(k_t) \quad (12)$$

We can easily deduce that $w'(k)k/w(k) = s(k)/\rho(k)$ and $R'(k)k/R(k) = -(1 - s(k))/\rho(k)$.

3 Equilibrium

3.1 Intertemporal equilibrium

Being given that the size of a young working generation equals one, the equilibrium in the labour market is ensured by $L_t = 1$. In addition, capital market clears, which requires that aggregate investment equals aggregate savings: $k_{t+1} = s_t$. From the budget constraint (3), this directly implies that $x_t = (1-\tau)w(k_t) - k_{t+1}$. Moreover, using (11), we can express the equilibrium levels of public services as:

$$\eta_t = \mu\tau w(k_t) \quad (13)$$
$$G_t = (1-\mu)\tau w(k_t) \quad (14)$$

Substituting (11) and (14) into equation (10), we obtain:

$$P_{t+1} = (1-m)P_t + \epsilon_1 f(k_t) - \epsilon_2(1-\mu)\tau w(k_t) \quad (15)$$

Then, we can rewrite equation (7) with equality as:

$$k_{t+1} = \frac{1+\theta_t}{1+\alpha\frac{\sigma}{1-\sigma}+\theta_t}(1-\tau)w(k_t) \quad (16)$$

with

$$\theta_t = \frac{[(1-\tau)w(k_t) - k_{t+1}]^{\alpha}[\mu\tau w(k_t)]^{1-\alpha}}{P_t^{\beta}} \quad (17)$$

Given $k_0 \geqslant 0$ and $P_0 > 0$, equations (15)-(17) define the dynamics of the economy through the evolution of the sequence (k_t, P_t), for all $t \geqslant 0$.

Before going further and derive our main results and in order to stick with reality, let us assume the following parametric hypothesis:

Assumption 2 $\epsilon_1 > \epsilon_2$

Assumption 2 is not so much restrictive as it means that pollution flows exceed abatement flows, reflecting what can be observed in real world. It also implies that the available technology of abatement is not so much efficient with respect to the emission rate of pollution per unit of production. Again, this seems to be consistent with empirical evidences.

Given the dynamic system (15)-(17), we can identify several mechanisms crucial to our analysis and our understanding of the dynamic behavior of the economy. At this stage, we have already underscored the endogenous character of the saving rate $\frac{1+\theta_t}{1+\alpha\frac{\sigma}{1-\sigma}+\theta_t}$ (see equation (16)). In particular, despite the *a priori* complementarity between private and public health expenditure, we have explained that a enhanced health indicator lowers the marginal return on private health spending, but in turn, fosters capital accumulation.

Then, we can emphasize two complementary components that also affect longevity and which are taken as given by households. Indeed, public health expenditure positively influence longevity, whereas pollution entails a negative externality. These two complementary components are mainly driven by the government behavior, that is the allocation of her resources between health care and pollution abatement. These two policies are targeted to reach a unique goal, which is a lengthening of lifetime. Nevertheless, note that the sequence of outcome is not simultaneous: A larger amount of health expenditure involves a longer life expectancy of the current generation while a more substantial effort of abatement displays its positive consequences for the next generation only.

We turn now to the analysis of the existence and uniqueness of the stationary equilibrium.

3.2 Stationary equilibrium

Using (15)-(17), a steady state is defined by $k_t = k_{t+1} = k$ and $P_t = P_{t+1} = P$, for all t, satisfying:

$$P = \frac{[(1-\tau)w(k)-k]^{\frac{\alpha+1}{\beta}}[\mu\tau w(k)]^{\frac{1-\alpha}{\beta}}}{\left[\left(\alpha\frac{\sigma}{1-\sigma}+1\right)k-(1-\tau)w(k)\right]^{\frac{1}{\beta}}} \equiv \varphi(k) \tag{18}$$

$$P = \frac{1}{m}[\epsilon_1 f(k) - \epsilon_2(1-\mu)\tau w(k)] \equiv \psi(k) \tag{19}$$

Using these two equations, we can demonstrate the existence and the uniqueness of the stationary solution (k^*, P^*).

Proposition 1 *Let $\overline{k}$ and $\underline{k}$ be defined by:*

$$\overline{k} = (1-\tau)w(\overline{k}) \tag{20}$$

$$\underline{k} = \frac{(1-\tau)w(\underline{k})}{1+\alpha\frac{\sigma}{1-\sigma}} \tag{21}$$

Under Assumptions 1-2, there exists a unique steady state $k^* > 0$ that belongs to $(\underline{k}, \overline{k}]$. This implies that there exists a unique value $P^* > 0$.

Proof. See Appendix B. ■

The stationary solution eventually reached by one economy crucially depends on the public policy implemented by the government. We tackle this issue in the following.

4 May public policy be welfare improving?

In this section, we study the consequences associated with a change in the policy design, that is a larger share of public revenue devoted to public health. In particular, we investigate whether such a policy may improve welfare W^* or not in the long term. Let us first define the stationary welfare as follows:

$$W \equiv \pi(\theta)\frac{c^{1-\sigma}}{1-\sigma} \tag{22}$$

As a preliminary result, we can show that the welfare at the steady state can be defined as an increasing function of physical capital only:

Lemma 2 *Under Assumptions 1-2, the welfare evaluated at the steady state W^* is an increasing function of capital, i.e. $W^* \equiv W(k^*)$ with $W'(k^*) > 0$.*

Proof. See Appendix C. ■

Indeed, using (1), (4) and (6), we can observe that utility is increasing with respect to longevity and remunerated savings. The last one increases with respect to capital. Using (16), we also deduce that the indicator of health depends positively on the stock of physical capital at the steady state. This implies that the richer the economy, the better the health status, despite the level of pollution. In other words, the increasing expenditure in private and public health always offset the damaging effect of a growing stock of pollution. This explains that welfare raises with capital accumulation.

The design of the public policy may explain why one economy reaches a steady state with larger levels of capital, pollution and/or welfare compared to another one. We study now more carefully the outcome induced by a change in the public policy design, that is a raise in μ, on these stationary levels of capital, welfare and pollution:

Proposition 2 *Under Assumptions 1-2, a change in the policy design in favor of a larger support to public health care, i.e. a larger μ, displays the following effects:*

1. *k^* and W^* are increasing in μ if and only if $(1-\alpha)[\epsilon_1 f(k^*) - \epsilon_2(1-\mu)\tau w(k^*)] > \beta\epsilon_2\mu\tau w(k^*)$, which is satisfied for $1-\alpha > \beta\frac{\epsilon_2\mu\tau}{\epsilon_1-\epsilon_2(1-\mu)\tau}$;*
2. *k^* and W^* are decreasing in μ if and only if $(1-\alpha)[\epsilon_1 f(k^*) - \epsilon_2(1-\mu)\tau w(k^*)] < \beta\epsilon_2\mu\tau w(k^*)$, which is satisfied for $1-\alpha < \beta\frac{\epsilon_2\mu\tau}{2\epsilon_1}$;*

In addition, pollution P^* is always increasing in μ.

Proof. See Appendix D. ■

When dealing with pollution, the raising share of public revenue devoted to public health is detrimental to the effort of public abatement and yields a clear-cut outcome: Pollution always raises. As for the welfare *per se* or the

stock of capital, conclusions are more ambiguous. They even depend on both the initial sharing of public receipts, the level of the tax and the stationary stock of physical capital. This being so, we can claim that two economies that share quite similar economic and technological features may nonetheless differ in the long run due to the implemented public policy. According to Proposition 2, in case 1, one economy that chooses to invest more in public health care should be better off in the long term. Conversely, in the second case, one economy that gives priority to environmental preservation should be wealthier. Why is it so? The reasoning behind this result is presented below.

We have explained that capital accumulation accelerates when health status (and longevity) enhances and that a larger value of μ triggers two opposite effects on health characteristics: A positive one through a larger amount of public health expenditure and a negative one because this simultaneously lowers public environmental protection, which involves more pollution. Proposition 2 states that the first (second) effect dominates when the weight of public health $1-\alpha$ in the health indicator is sufficiently large (low) relative to the weight of pollution β. So, when public health is a major component of health status, the noxious effect of a growing pollution stock is overweighted by a greater improvement in health status. Overall, the welfare is enhanced at the steady state. On the contrary, when the role played by health determinants is relatively minor, a public policy in favor of health displays a stronger negative effect on the quality of the environment, which annihilates its positive effect on life expectancy and capital accumulation.

Beyond this statement, we may also emphasize the role played by the initial public finances allocation. In particular, if the sharing rule is already beneficial towards public health, then, configuration 2 is more likely. Hence, there is less need of reinforcing it, otherwise it can be costly in terms of wealth accumulation and welfare. Conversely, for a relatively low initial value of μ, configuration 1 is more likely. This might be explained thanks to the concavity property of the longevity function with respect to the health indicator. A more generous allocation of public funds towards health has a stronger positive effect on longevity when it is low enough, thus promoting fast capital accumulation. On the contrary, the marginal gain of promoting public health in terms of longevity is negligible when it is already high. But, the subsequent increase in pollution (due to a shift in public fund allocation) is relatively more damaging, and this might reduce lifetime length. Therefore, if the relationship between the capital stock and the sharing rule of public funds seems to be non-monotonous, there might be a room for determining the second best optimal policy that maximises welfare at the steady state.

Having this in mind, we note that Proposition 2 gives necessary and sufficient conditions as well as sufficient conditions to evaluate the consequences of a change in μ on stationary capital and welfare. While the last ones do not depend on the steady state k^*, the necessary and sufficient conditions do. Therefore, it looks interesting to focus on the case of a Cobb-Douglas technology where more clear-cut conclusions can be obtained.

Corollary 1 *Let*

$$\widehat{\mu} \equiv \frac{(1-\alpha)(\epsilon_1 - \epsilon_2\tau(1-s))}{\epsilon_2\tau(1-s)(\beta-(1-\alpha))} \tag{23}$$

Under Assumptions 1-2, assume further that $f(k) \equiv Ak^s$*, with* $A > 0$ *and* $0 < s < 1/2$*.*

1. *If* $\beta \leqslant (1-\alpha)\epsilon_1/[\epsilon_2\tau(1-s)]$*,* k^* *and* W^* *are increasing in* μ *for all* $\mu \in (0,1]$*.*
2. *If* $\beta > (1-\alpha)\epsilon_1/[\epsilon_2\tau(1-s)]$*,* k^* *and* W^* *are increasing in* μ *for all* $\mu \in (0,\widehat{\mu}]$ *and decreasing in* μ *for all* $\mu \in [\widehat{\mu},1]$*.*

Proof. See Appendix E. ■

The first result of this corollary confirms Proposition 2. When longevity is barely sensitive to pollution compared to usual health components, a larger share of public spending devoted to public health always induces a higher level of capital and welfare, through its positive effect on longevity, which in turns fosters savings. From a normative point of view, the optimal policy design boils down to set $\mu = 1$.

The second result is even more of interest. It clearly highlights the envisioned inverse U-shaped relationship between the policy design μ and the economic variables k^* or W^*. In a world of second best optimality, there exists a level of public spending devoted to public health $\hat{\mu}$, as defined in Corollary 1, that maximizes both the stationary welfare and level of physical capital. We argue that this may still be justified by the two opposite effects induced by a shift in the value of μ on the health status and therefore, on longevity, as described above.

5 The destabilizing role of pollution

In this section, we analyse the convergence of the economy towards the previously identified steady state (see Proposition 1). We show that the complementarity between pollution and (public and private) health that characterizes longevity may be a source of instability and endogenous cycles.

For ease of presentation, we establish that choosing an appropriate value of $m \in (0,1)$ allows us to normalize the steady state k^*, such that $k^* = 1$. Then, we lead the analysis of the dynamics studying the stability properties of the steady state.

Proposition 3 *Let*

$$\overline{\epsilon}_1 \equiv \frac{[(1-\tau)w(1)-1]^{\frac{\alpha+1}{\beta}}[\mu\tau w(1)]^{\frac{1-\alpha}{\beta}}}{f(1)\left(\frac{\alpha\sigma}{1-\sigma}+1\right)^{1/\beta}} \tag{24}$$

Assuming that $\epsilon_1 < \bar{\epsilon}_1$[9], $1 < (1-\tau)w(1) < 1+\alpha\frac{\sigma}{1-\sigma}$ *and Assumptions 1-2 hold, there exists a unique* $m^* \in (0,1)$ *that ensures that* $k^* = 1 \in (\underline{k}, \overline{k})$.

Proof. See Appendix F. ■

We deduce the level of pollution that comes along with the normalized steady state $k^* = 1$:

$$P^* = \frac{\left[(1-\tau)w-1\right]^{\frac{\alpha+1}{\beta}}\left[\mu\tau w\right]^{\frac{1-\alpha}{\beta}}}{\left[\alpha\frac{\sigma}{1-\sigma}+1-(1-\tau)w\right]^{\frac{1}{\beta}}}, \tag{25}$$

where we denote $w \equiv w(1)$, meanwhile we define $s \equiv s(1)$ and $\rho \equiv \rho(1)$. To analyse the stability properties of the steady state, we differentiate the dynamic system (15)-(16) around the normalized steady state. We establish the following:

Lemma 3 *Assuming that* $\epsilon_1 < \bar{\epsilon}_1$, $1 < (1-\tau)w < 1+\alpha\frac{\sigma}{1-\sigma}$ *and Assumptions 1-2 hold, the characteristic polynomial evaluated at the steady state* $k^* = 1$ *is given by* $P(\lambda) = \lambda^2 - T(\rho)\lambda + D(\rho) = 0$, *where:*

$$T(\rho) = 1 - m^* + \frac{s}{\rho}\frac{1+\frac{(1-\tau)w-(1-\alpha)}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}} \tag{26}$$

$$\begin{aligned} D(\rho) &= (1-m^*)\frac{s}{\rho}\frac{1+\frac{(1-\tau)w-(1-\alpha)}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}} \\ &+ m^*\frac{s}{\rho}\frac{\rho\epsilon_1-\epsilon_2(1-\mu)\tau(1-s)}{\epsilon_1-\epsilon_2(1-\mu)\tau(1-s)}\frac{\frac{\beta\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}} \end{aligned} \tag{27}$$

and

$$\theta = \frac{\alpha\sigma/(1-\sigma)+1-(1-\tau)w}{(1-\tau)w-1} \tag{28}$$

Proof. See Appendix G. ■

We now determine the stability properties of the steady state, *i.e.* whether it is a saddle, a source or a sink. Since the two dynamic variables k_t and P_t are predetermined, the dynamic path locally converges to the steady state only if it is a sink. We also focus on the existence of endogenous cycles studying the occurrence of bifurcations. To simplify our proofs, we assume:

Assumption 3 $s \geqslant 1/3$.

This parametric assumption means that $2s \geqslant 1-s$ and according to Assumption 1, $\rho \in (2s, +\infty)$. Then, we can establish a first result on the stability properties of the steady state:

[9] Of course, this last inequality may be compatible with Assumption 2.

Lemma 4 *Assuming that* $\epsilon_1 < \bar{\epsilon}_1$, $1 < (1-\tau)w < 1 + \alpha\frac{\sigma}{1-\sigma}$ *and Assumptions 1-3 hold, the steady state can never be a saddle. It is either a sink or a source.*

Proof. See Appendix H. ■

This lemma shows that starting in a neighbourhood of the steady state, the economy either converges to this equilibrium or there is instability. Choosing the elasticity of capital-labour substitution ρ as a bifurcation parameter, we clarify whenever one of these two configurations occurs. We discuss the results according to the level of β, *i.e.* the weight of pollution in the health indicator. Indeed, as mentioned in Introduction, the role played by environmental factors in the worldwide evolution of life expectancy has sharply increased over the last decades in comparison with usual health determinants. This phenomenon can be formally captured by the parameter β and the different values it can takes. A larger value of the parameter β means of course that the economy is more vulnerable to environmental risks.

Proposition 4 *Assuming that* $\epsilon_1 < \bar{\epsilon}_1$, $1 < (1-\tau)w < 1 + \alpha\frac{\sigma}{1-\sigma}$ *and Assumptions 1-3 hold, there exist* $A_\beta > 0$, $0 < B_\beta < 1$, $C_\beta > 0$ *and* $\Delta_\beta > 0$, *such that:*

1. *If* β *is sufficiently low, satisfying* $\beta < \min\{1/A_\beta; (1-B_\beta)/C_\beta\}$, *the steady state is a sink for all* $\rho \in (2s, +\infty)$;
2. *If* $\mu < 1 - \epsilon_1 s/[\epsilon_2\tau(1-s)]$[10], α *sufficiently low and* β *larger but close to* $1/A_\beta$, *there exists* $\rho_H \in (2s, +\infty)$ *such that the steady state is a sink for* $\rho \in (2s, \rho_H)$, *a Hopf bifurcation generically occurs for* $\rho = \rho_H$, *and the steady state is a source for* $\rho \in (\rho_H, +\infty)$;
3. *If* μ *is close to 1 and* β *smaller but close to* $1/A_\beta$, *there exists* $\rho_H \in (2s, +\infty)$ *such that the steady state is a source for* $\rho \in (2s, \rho_H)$, *a Hopf bifurcation generically occurs for* $\rho = \rho_H$, *and the steady state is a sink for* $\rho \in (\rho_H, +\infty)$;
4. *If* β *is sufficiently large, satisfying* $\beta > \max\{1/A_\beta; (1-B_\beta)/C_\beta\}$, *the steady state is a source for all* $\rho \in (2s, +\infty)$.

In addition, when $\beta < \Delta_\beta$ *the eigenvalues are real, whereas when* $\beta > \Delta_\beta$, *the eigenvalues are complex conjugates.*

Proof. See Appendix I. ■

When pollution weakly affects longevity (β sufficiently low), the economy converges to the steady state. If β is even lower than Δ_β, the eigenvalues are real and the convergence is monotonic. On the contrary, if β satisfies $\beta > \Delta_\beta$, one converges to the steady state through oscillations.

[10]Note that according to Assumptions 1-3, this inequality may be satisfied only if τ is sufficiently large and ϵ_2 sufficiently close to ϵ_1, that is $\tau > \epsilon_1 s/[\epsilon_2(1-s)]$.

On the contrary, when pollution is considered to be a driving factor of longevity (β sufficiently large), the steady state turns out to be unstable. Of course, according to our previous comment, if β satisfies $\beta > \Delta_\beta$, this divergence also experiences unstable oscillations.

These two cases show that non-monotonic oscillations and instability occur as soon as pollution strongly affects longevity. Therefore, environmental quality, and its complementarity with health in longevity, is a key ingredient to explain non-monotonic or even unstable dynamic path. However, between these two cases, there are two additional interesting configurations.

For either values of μ and α sufficiently low, or μ sufficiently close to 1, there exists a level of the capital-labor elasticity of substitution such that there is a change in the stability properties (between between sink and source). Then, an endogenous cycle occurs, through a Hopf bifurcation. This happens when the weight of pollution in the health indicator takes intermediate values, *i.e.* for β arbitrarily close to $1/A_\beta$.

To grab the economic intuition for the existence of these non-monotonic dynamic paths, the reader should recall that the dynamics are determined by (15)-(16), taking into account that θ_t is given by (17). We argue that the explanation of oscillations and endogenous cycles mainly derives from the complementarity between health, private and public ones, and pollution in the health indicator θ_t. The higher the health status, the longer the lifetime, and therefore, the larger the saving rate $(1+\theta_t)/(1+\alpha\frac{\sigma}{1-\sigma}+\theta_t)$, whereas deteriorated health conditions may induce a reversal of capital accumulation, because of a lower saving rate.

Consider first that β is arbitrarily close to 0 so that the effect of pollution on health is negligible. We can easily highlight the mechanisms at stake that induce a positive relationship between k_t and k_{t+1}, which then implies a monotonic dynamics and excludes any oscillations. Indeed, following a raise in physical capital, k_t, two effects arise. An income effect that triggers both private health expenses and savings, and a longevity effect that reinforces the positive effect on savings. Indeed, beyond the income effect, a raise in k_t simultaneously drives up the provision of public health care, which in turns improves the health indicator and longevity. Hence, incentives to invest in private health are reduced in favor of physical capital accumulation, because the marginal return on private health investment is lower. Hence, a raise in k_t always supports larger savings and thus physical capital accumulation, which also explains monotonic convergence.

Assume now that β is large enough. Pollution strongly affects the health status and there exists a complementarity between health, be it private and public, and environmental quality. Besides, the stock of pollution P_t shapes longevity of households born at date t, but positively depends on past stock of physical capital k_{t-1}. Consider now a sequence of physical capital such that k_{t-1} and k_t raise through time. Using (16), we deduce that of course the income raises since k_t increases, but now two opposite effects on health occur. On the one hand, the increase of physical capital by augmenting public healthcare provision pushes up the saving rate. On the other hand, an increase of k_{t-1} induces more pollution, which is harmful for the health indicator and, therefore, for savings. Because of this last feedback effect, a sequence of growing capital

stock may experience a reversal, which explains the existence of oscillations in this model. Hence, in one economy where environmental factors become crucial determinants of life expectancy relative to usual health components, the stability of the economy is threatened. This is a serious concern to be considered by public authorities, as in many recent reports the WHO has underlined the growing role played by pollution in the evolution of longevity. We could even argue that in the case where β is very large, the sustainability of the growth process is reconsidered: It appears that pollution generates some irreversibility to that extent that it prevents the economy from reaching the steady state but rather generates structural instability.

Proposition 4 further shows that persistent endogenous cycles occur through a Hopf bifurcation if either μ and α are close to 0 or μ close to 1. Indeed, in both cases, the feedback effect of pollution is reinforced and the sensitivity of the economy to pollution is increased. When μ is low, public spendings are mainly devoted to pollution abatement and mechanically the provision of public health care is drastically low. Since α is also small, private health also displays a weak effect on the health indicator. When μ is close to 1, public expenditure are mainly dedicated to public health. Obviously, public abatement is negligible and the positive effect of k_{t-1} on the stock of pollution is well much stronger. All together, these interactions involve endogenous cycles that might compromise sustainability in the long term since the economy never attain the stationary solution. This kind of result has been also highlighted by Zhang (1999), although the sources of these fluctuations are not quite similar. Here, it is mainly due to the apparently inappropriate public policy design, which in fact intensifies the negative pollution externality.

6 Stabilisation versus stationary welfare

In Section 4, we have extensively discussed the effects of the public policy on the stationary welfare. We now examine whether these results are mitigated or not by the consequences of the same public policy on the stability properties of the steady state.

Focusing on the most interesting configurations where endogenous cycles can occur, *i.e.* cases 2 and 3 of Proposition 4, we investigate whether a variation of the policy parameter μ is able to rule out endogenous cycles and restore stability.

Proposition 5 *Assuming that* $\epsilon_1 < \bar{\epsilon}_1$, $1 < (1-\tau)w < 1 + \alpha\frac{\sigma}{1-\sigma}$ *and Assumptions 1-3 hold, the following holds.*

1. *If* $\mu < 1 - \epsilon_1 s/[\epsilon_2\tau(1-s)]$, α *sufficiently low and* β *larger but arbitrarily close to* $1/A_\beta$, *a slight increase of* μ *can rule out endogenous cycles, because* β *becomes lower than* $1/A_\beta$. *Then, the steady state is stable for all* $\rho \in (2s, +\infty)$.

2. *If* μ *is close to 1 and* β *smaller but arbitrarily close to* $1/A_\beta$, *a slight*

decrease of μ can rule out endogenous cycles, because β becomes larger than $1/A_\beta$. Then, the steady state is unstable for all $\rho \in (2s, +\infty)$.

Proof. See Appendix J. ■

We have previously given sufficient conditions for the occurrence of a Hopf bifurcation. We have also highlighted that endogenous cycles can emerge when the share of public spending devoted to public health μ is either sufficiently low (configuration 2 of Proposition 4) or sufficiently close to 1 (configuration 3 of Proposition 4). This corollary shows that if initially public funds are mainly dedicated to environmental protection (μ is low), then a slight increase of μ may rule out these cycles; Whereas if μ is initially close to 1, a slight decrease of μ may eliminate them. Therefore, depending on whether the share of public spending devoted to public health care is either low or large and close to 1, an increase or a decrease of μ is recommended to eliminate cycles. In both configurations, the change in the policy design mitigates the feedback effects induced by the accumulation of pollution.

However, there is a main difference between these two results. If μ is low, a slight increase of μ induces stability. If μ is close to 1, a slight decrease of μ creates instability for all $\rho \in (2s, +\infty)$, whereas before, there was instability for $\rho \in (2s, \rho_H)$ only. This also means that starting with instability for all $\rho \in (2s, +\infty)$, a slight increase of μ may be a source of stability for $\rho \in (\rho_H, +\infty)$. Hence, even for μ sufficiently close to 1, an increase of μ can promote stability for a non-empty range of elasticities of capital-labour substitution ρ. For a given vulnerability of life expectancy to pollution, it seems possible to eliminate cycles by choosing an appropriate public policy. Nevertheless, the allocation of public funds should be cautiously used since it could be the case that it fosters the occurrence of aggregate instability rather than promotes a stable dynamic path. The following proposition summarizes our conclusions.

Proposition 6 *Assume that $\epsilon_1 < \overline{\epsilon}_1$, $1 < (1-\tau)w < 1+\alpha\frac{\sigma}{1-\sigma}$ and Assumptions 1-3 hold. If β larger but arbitrarily close to $1/A_\beta$ and either $\mu < 1-\epsilon_1 s/[\epsilon_2\tau(1-s)]$ and α sufficiently low, or μ close to 1, a slight increase of μ has a stabilizing effect by reducing the range of ρ for instability.*

It is necessary to investigate the outcome of such a policy design in the light of the analysis lead in Section 4. In particular, we wonder whether this result is consistent or compatible with the policy recommendations established at the steady state. Indeed, we have demonstrated (see Proposition 2) that a larger support towards public health is welfare enhancing if μ is initially low enough. The results we obtain in this section reinforce this suggestion, underlying that it can also stabilize the economy. For μ sufficiently close to 1, the conclusions are mitigated. Even if a raise of μ can bring forward economic stability - for some values of ρ - it can also reduce the stationary welfare. Indeed, configuration 2 of Proposition 2 is more likely in this case. Therefore, the stabilizing policy may be detrimental for welfare, while for μ rather low, it can satisfy both the economic stability and the improvement of the stationary equilibrium.

7 Conclusion

This paper presents an overlapping generations model where longevity is endogenously influenced by public health provision, pollution and private health efforts undertaken by individuals. Public expenditure, financed thanks to labour taxation, provide both public health and abatement. A larger support towards public health may raise the welfare evaluated at the unique steady state, if initially the sharing rule of public fund is more favorable to pollution abatement and/or if pollution has a negligible impact on longevity. In contrast, if initially, the amount of public heath expenses is already large and/or if pollution is a crucial determinant of longevity, increasing the effort of abatement is recommended to improve welfare. As a warning and a recommendation for policy makers, we show that the complementarity between pollution and health in the longevity can also be source of aggregate economic instability: When the effect of pollution on longevity is large enough, endogenous cycles may occur and the steady state becomes unstable. Then, we underline the possible stabilizing effect of a fiscal receipts allocation that would increase the share of public spending devoted to public health. However, we mitigate our conclusions since such a fiscal policy may deteriorate capital accumulation and welfare at the steady state, if initial public health expenses almost cover public spending.

8 Appendices

A Proof of Lemma 1

First of all, to prove Lemma 1, we can easily show that $\lim_{x_t \to 0} \theta_t = 0$. Let us note $\Gamma_t > 0$ the left-hand side of inequality (7), *i.e.* $\Gamma_t \equiv \frac{1}{1+\theta_t} \frac{\alpha}{x_t}$. Then, $\lim_{x_t \to 0} \Gamma_t = +\infty$. Therefore, $\Gamma_t < \frac{1-\sigma}{\sigma s_t}$ cannot hold for $x_t = 0$, which implies that $x_t > 0$ and $\Gamma_t = \frac{1-\sigma}{\sigma s_t}$.

B Proof of Proposition 1

Any steady state should satisfy $P \geqslant 0$. Under Assumption 2, $P = \psi(k) \geqslant 0$ for all $k \geqslant 0$. In contrast, $P = \varphi(k) \geqslant 0$ requires:

$$w(k)/k \geqslant 1/(1-\tau) \tag{A.1}$$

$$\left(\alpha \frac{\sigma}{1-\sigma} + 1\right)/(1-\tau) > w(k)/k \tag{A.2}$$

Since $w(k)/k = f(k)/k - f'(k)$, we can easily show that $\lim_{k \to +\infty} w(k)/k = 0$. From Assumption 1, there exist values of $k > 0$ such that $w(k)/k > \left(\alpha \frac{\sigma}{1-\sigma} + 1\right)/(1-\tau)$. Since $d\ln[w(k)/k]/d\ln k = s(k)/\rho(k) - 1 < 0$, we also note that $w(k)/k$ is decreasing. This shows the existence of $\overline{k}$ and $\underline{k}$, defined by (20) and (21) respectively, such that $\underline{k} < \overline{k}$. Any $k \in (\underline{k}, \overline{k}]$ satisfies inequalities (A.1) and (A.2).

We show now the existence of a steady state that belongs to $(\underline{k}, \overline{k}]$. By direct inspection of (18), we deduce that $\varphi(\underline{k}) = +\infty$ and $\varphi(\overline{k}) = 0$. Moreover,

$$\begin{aligned}\psi(\underline{k}) &< \frac{1}{m}\epsilon_1 f(\underline{k}) < +\infty \\ \psi(\overline{k}) &> \frac{1}{m} f(\overline{k})[\epsilon_1 - \epsilon_2(1-\mu)\tau] > 0\end{aligned}$$

By continuity, there is at least one solution $k^* \in (\underline{k}, \overline{k})$ solving $\varphi(k^*) = \psi(k^*)$.

To show uniqueness, let us note $\epsilon_\varphi(k) \equiv \varphi'(k)k/\varphi(k)$ and $\epsilon_\psi(k) \equiv \psi'(k)k/\psi(k)$. Using (18) and (19), we get:

$$\epsilon_\psi(k) = \frac{s(k)}{\rho(k)} \frac{\epsilon_1\rho(k) - \epsilon_2(1-\mu)\tau(1-s(k))}{\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s(k))} \tag{A.3}$$

$$\begin{aligned}\epsilon_\varphi(k) = {} & \frac{1}{\beta}\frac{2(1-\tau)w(k)s(k)/\rho(k) - [1+\alpha+(1-\alpha)s(k)/\rho(k)]k}{(1-\tau)w(k) - k} \\ & - \frac{1}{\beta}\frac{[1+\alpha\sigma/(1-\sigma)]k - (1-\tau)w(k)s(k)/\rho(k)}{[1+\alpha\sigma/(1-\sigma)]k - (1-\tau)w(k)}\end{aligned} \tag{A.4}$$

Under Assumptions 1 and 2, $\epsilon_\psi(k) > 0$. Moreover, the first term in (A.4) is lower than $1/\beta$, whereas the second one is larger than $1/\beta$. We conclude that $\epsilon_\varphi(k) < 0$, which shows the uniqueness of the steady state.

C Proof of Lemma 2

In the long run, the welfare W is defined by $W \equiv \pi(\theta)c^{1-\sigma}/(1-\sigma)$. Since consumption is given by $c = R(k)k/\pi(\theta)$, the welfare may be rewritten:

$$W = \pi(\theta)^\sigma (R(k)k)^{1-\sigma}/(1-\sigma) \tag{A.5}$$

Using (18), we have:

$$\theta = \frac{[(1-\tau)w(k) - k]^\alpha [\mu\tau w(k)]^{1-\alpha}}{P^\beta} = \frac{\left(\alpha\frac{\sigma}{1-\sigma} + 1\right)k - (1-\tau)w(k)}{(1-\tau)w(k) - k} \equiv \theta(k) \tag{A.6}$$

Substituting $\theta = \theta(k)$ into (A.5), the welfare becomes a function of k, namely $W \equiv W(k)$. Therefore, at the steady state k^*, the welfare is given by $W(k^*)$. Using Assumption 1, $R(k)k$ is increasing in k. We also know that $\pi(\theta)$ is increasing in θ. Therefore, $W(k^*)$ is an increasing function of physical capital if $\theta'(k) > 0$. Using (A.6), we get:

$$\frac{\theta'(k)k}{\theta(k)} = \frac{\left(\alpha\frac{\sigma}{1-\sigma} + 1\right)k - (1-\tau)w(k)\frac{s(k)}{\rho(k)}}{\left(\alpha\frac{\sigma}{1-\sigma} + 1\right)k - (1-\tau)w(k)} - \frac{(1-\tau)w(k)\frac{s(k)}{\rho(k)} - k}{(1-\tau)w(k) - k} > 0$$

because, according to Assumption 1, $\rho(k) > s(k)$.

D Proof of Proposition 2

Let us note $\epsilon_\varphi(\mu) \equiv \frac{\partial \varphi(k)}{\partial \mu}\frac{\mu}{k}$ and $\epsilon_\psi(\mu) \equiv \frac{\partial \psi(k)}{\partial \mu}\frac{\mu}{k}$. The steady state k^* is given by $\varphi(k^*) = \psi(k^*)$. Differentiating this equation with respect to k and μ, we get:

$$\frac{dk^*}{d\mu}\frac{\mu}{k^*} = \frac{\epsilon_\varphi(\mu) - \epsilon_\psi(\mu)}{\epsilon_\psi(k^*) - \epsilon_\varphi(k^*)} \tag{A.7}$$

We have $\epsilon_\psi(k^*) > \epsilon_\varphi(k^*)$. Therefore, the sign of $\frac{dk^*}{d\mu}\frac{\mu}{k^*}$ is given by $\epsilon_\varphi(\mu) - \epsilon_\psi(\mu)$. Using (18) and (19), we have:

$$\epsilon_\varphi(\mu) = \frac{1-\alpha}{\beta} \tag{A.8}$$

$$\epsilon_\psi(\mu) = \frac{\epsilon_2 \mu \tau w(k^*)}{\epsilon_1 f(k^*) - \epsilon_2(1-\mu)\tau w(k^*)} \tag{A.9}$$

We easily deduce the necessary and sufficient conditions to have either $dk^*/d\mu > 0$ or $dk^*/d\mu < 0$. We now determine sufficient conditions that do not depend on the steady state level k^*.

One one hand, we note that $\epsilon_\psi(\mu) < \frac{\epsilon_2 \mu \tau f(k^*)}{[\epsilon_1 - \epsilon_2(1-\mu)\tau] f(k^*)} = \frac{\epsilon_2 \mu \tau}{\epsilon_1 - \epsilon_2(1-\mu)\tau}$. Using (A.8) and (A.9), we deduce that $\epsilon_\varphi(\mu) - \epsilon_\psi(\mu) > 0$ if $1-\alpha > \beta \frac{\epsilon_2 \mu \tau}{\epsilon_1 - \epsilon_2(1-\mu)\tau}$.

On the other hand, $\epsilon_\psi(\mu) > \frac{\epsilon_2 \mu \tau w(k^*)}{\epsilon_1 f(k^*)} = \frac{\epsilon_2 \mu \tau (1-s(k^*))}{\epsilon_1} > \frac{\epsilon_2 \mu \tau}{2\epsilon_1}$ under Assumption 1. We deduce that $\epsilon_\varphi(\mu) - \epsilon_\psi(\mu) < 0$ if $1-\alpha < \beta \frac{\epsilon_2 \mu \tau}{2\epsilon_1}$.

Of course, using Lemma 2, we deduce the effects of μ on the stationary welfare W^*. We focus now on the effects induced by a change in μ on pollution P^*. Using (19) and (A.7), we have:

$$\begin{aligned} \frac{dP^*}{d\mu}\frac{\mu}{P^*} &= \epsilon_\psi(k^*)\frac{dk}{d\mu}\frac{\mu}{k^*} + \epsilon_\psi(\mu) \\ &= \frac{\epsilon_\psi(k^*)\epsilon_\varphi(\mu) - \epsilon_\varphi(k^*)\epsilon_\psi(\mu)}{\epsilon_\psi(k^*) - \epsilon_\varphi(k^*)} \end{aligned}$$

Since $\epsilon_\psi(k^*) > 0$, $\epsilon_\varphi(k^*) < 0$, $\epsilon_\varphi(\mu) > 0$ and $\epsilon_\psi(\mu) > 0$, we easily deduce that $\frac{dP^*}{d\mu}\frac{\mu}{P^*} > 0$.

E Proof of Corollary 1

First note that $f(k) = Ak^s$, with $A > 0$ and $s \in (0, 1/2)$, satisfies Assumption 1. Using this production function, we have $w(k^*)/f(k^*) = 1 - s$. Therefore, using the proof of Proposition 2, $\epsilon_\varphi(\mu) > \epsilon_\psi(\mu)$ if and only if:

$$(1-\alpha)[\epsilon_1 - \epsilon_2\tau(1-s)] > \mu\epsilon_2\tau(1-s)[\beta - (1-\alpha)]$$

Since $\mu \in (0, 1]$, this inequality is satisfied if $\beta \leqslant (1-\alpha)\epsilon_1/[\epsilon_2\tau(1-s)]$ or $\beta > (1-\alpha)\epsilon_1/[\epsilon_2\tau(1-s)]$ and $\mu < \widehat{\mu}$. On the contrary, when $\beta > (1-\alpha)\epsilon_1/[\epsilon_2\tau(1-s)]$ and $\mu > \widehat{\mu}$, we obtain $\epsilon_\varphi(\mu) < \epsilon_\psi(\mu)$. Then, the proof of Proposition 2 allows us to deduce the corollary.

F Proof of Proposition 3

Using (18) and (19), there exists a unique value $m = m^*$ solving $\varphi(1) = \psi(1)$, with:

$$m^* = [\epsilon_1 f(1) - \epsilon_2(1-\mu)\tau w(1)] \frac{\left[\alpha\frac{\sigma}{1-\sigma} + 1 - (1-\tau)w(1)\right]^{\frac{1}{\beta}}}{[(1-\tau)w(1)-1]^{\frac{\alpha+1}{\beta}}[\mu\tau w(1)]^{\frac{1-\alpha}{\beta}}} \tag{A.10}$$

Of course, $m^* > 0$, whereas

$$m^* < \frac{\epsilon_1 f(1)\left(\alpha\frac{\sigma}{1-\sigma}+1\right)^{\frac{1}{\beta}}}{[(1-\tau)w(1)-1]^{\frac{\alpha+1}{\beta}}[\mu\tau w(1)]^{\frac{1-\alpha}{\beta}}} < 1$$

for $\epsilon_1 < \bar{\epsilon}_1$. Finally, we clarify that $k^* = 1$ belongs to $(\underline{k}, \overline{k})$ for $1 < (1-\tau)w(1) < 1 + \alpha\frac{\sigma}{1-\sigma}$.

G Proof of Lemma 3

We differentiate the dynamic system (15)-(16) around the normalized steady state $k^* = 1$. We get:

$$\begin{aligned}
\frac{dP_{t+1}}{P^*} &= (1-m^*)\frac{dP_t}{P^*} + m^*\frac{s}{\rho}\frac{\rho\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)}{\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)}\frac{dk_t}{k^*} \\
\frac{dk_{t+1}}{k^*} &= -\frac{\frac{\beta\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}\frac{dP_t}{P^*} \\
&+ \frac{s}{\rho}\frac{1+\frac{(1-\tau)w-(1-\alpha)}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}\frac{dk_t}{k^*}
\end{aligned}$$

where θ is given by (28). We deduce the trace $T(\rho)$ and the determinant $D(\rho)$ of the associated Jacobian matrix, given by (26) and (27) respectively.

H Proof of Lemma 4

The steady state is a saddle if either $P(-1) < 0 < P(1)$ or $P(1) < 0 < P(-1)$. Since $T(\rho) > 0$ and $D(\rho) > 0$, we have $P(-1) = 1 + T(\rho) + D(\rho) > 0$. To determine the sign of $P(1) = 1 - T(\rho) + D(\rho)$, let us note:

$$\Lambda(\rho) \equiv \frac{s}{\rho}\frac{1+\frac{(1-\tau)w-(1-\alpha)}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}} \tag{A.11}$$

Because $\Lambda(\rho)$ is decreasing in ρ and $\rho \in (2s, +\infty)$, we have $\Lambda(\rho) < \Lambda(2s)$. One can easily prove that $\Lambda(2s) < 1$, that shows that $\Lambda(\rho) < 1$ for all $\rho \in (2s, +\infty)$.

Using (26), (27) and (A.11), we deduce that:

$$\begin{aligned} P(1) &= m^*(1-\Lambda(\rho)) \\ &+ m^* \frac{s}{\rho} \frac{\rho\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)}{\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)} \frac{\frac{\beta\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}} > 0 \end{aligned}$$

Since $P(1) > 0$ and $P(-1) > 0$, the steady state cannot be a saddle, but is either a source or a sink.

I Proof of Proposition 4

Let

$$A_\beta \equiv \frac{m^* s\epsilon_1}{\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)} \frac{\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}} \tag{A.12}$$

$$B_\beta \equiv (1-m^*)\Lambda(2s) < 1 \tag{A.13}$$

$$C_\beta \equiv \frac{m^*}{2} \frac{2s\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)}{\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)} \frac{\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}} \tag{A.14}$$

$$\begin{aligned} \Delta_\beta \equiv & \left[1 - m^* - \frac{s}{\rho}\frac{1+\frac{1-\tau)w-(1-\alpha)}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1+\frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}\right]^2 \frac{\rho}{4m^*s} \\ & \frac{\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)}{\rho\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)} \left[\frac{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma)}{\theta\alpha\sigma/(1-\sigma)} + \frac{\alpha}{(1-\tau)w-1}\right] \end{aligned} \tag{A.15}$$

where m^* given by (A.10) is a function of β.

As a preliminary remark, by direct inspection of (A.12)-(A.15), we observe that A_β, B_β, C_β and Δ_β have finite and strictly positive values. In addition, we have $B_\beta < 1$. Using (28), we note that θ does not depend on β. On the contrary, m^* depends on β, but always belongs to $(0,1)$ (see Appendix F). Therefore, there exist values of β such that it may be larger or smaller than $1/A_\beta$, $(1-B_\beta)/C_\beta$ and Δ_β, respectively.

Using Proposition 3, we easily prove that $T^2(\rho) - 4D(\rho) < 0$ if and only if $\beta > \Delta_\beta$. In this case, the two eigenvalues are complex conjugates. On the contrary, $\beta < \Delta_\beta$ is equivalent to $T^2(\rho) - 4D(\rho) > 0$ and the two eigenvalues are real.

Taking into account the results of Lemma 4, the steady state is a sink if $D(\rho) < 1$ and is a source if $D(\rho) > 1$. Moreover, choosing $\rho \in (2s, +\infty)$ as a bifurcation parameter, a Hopf bifurcation generically occurs if $D(\rho)$ crosses 1 for a value of ρ that belongs to $(2s, +\infty)$.

By direct inspection of (27), we immediately see that $D(\rho)$ either monotonically increases or decreases with respect to ρ. We have:

$$D(+\infty) = A_\beta\beta \tag{A.16}$$

$$D(2s) = C_\beta\beta + B_\beta \tag{A.17}$$

Both $D(+\infty)$ and $D(2s)$ belong to $(0,1)$ when β is low enough. Since $D(\rho)$ monotonically varies with respect to ρ, the steady state is a sink for all $\rho \in (2s, +\infty)$. When β is sufficiently large, both $D(+\infty)$ and $D(2s)$ are larger than 1. In this case, the steady state is a source for all $\rho \in (2s, +\infty)$.

We now investigate whether there may be a change of stability through the occurrence of a Hopf bifurcation when ρ describes $(2s, +\infty)$. Using (A.16) and (A.17), $D(+\infty) > 1$ and $D(2s) < 1$ if and only if:

$$A_\beta \beta > 1 \text{ and } C_\beta \beta + B_\beta < 1 \tag{A.18}$$

Taking $\beta = 1/A_\beta$, the second inequality rewrites $C_\beta/A_\beta + B_\beta < 1$. Using (A.12) and (A.14),

$$\frac{C_\beta}{A_\beta} = \frac{2s\epsilon_1 - \epsilon_2(1-\mu)\tau(1-s)}{2s\epsilon_1} < \frac{1}{2} \tag{A.19}$$

if and only if $\mu < 1 - \epsilon_1 s/[\epsilon_2 \tau(1-s)]$. Moreover, using (A.11) and (A.13), $\Lambda(2s)$ tends to $1/2$ when α tends to 0, which ensures $B_\beta < 1/2$. By continuity, for α low enough and β larger but sufficiently close to $1/A_\beta$, the two inequalities in (A.18) are satisfied. Therefore, we can conclude that there exists $\rho_H \in (2s, +\infty)$ satisfying $D(\rho_H) = 1$. Moreover, $D(\rho) < 1$ for $\rho \in (2s, \rho_H)$, whereas $D(\rho) > 1$ for $\rho \in (\rho_H, +\infty)$.

Finally, we have $D(+\infty) < 1$ and $D(2s) > 1$ if and only if:

$$A_\beta \beta < 1 \text{ and } C_\beta \beta + B_\beta > 1 \tag{A.20}$$

Taking $\beta = 1/A_\beta$, the second inequality rewrites $C_\beta/A_\beta + B_\beta > 1$. Using (A.19), we see that C_β/A_β is close to 1 for μ close to 1. Therefore, for β smaller but sufficiently close to $1/A_\beta$, the two inequalities in (A.20) are satisfied. We can conclude that there exists $\rho_H \in (2s, +\infty)$ satisfying $D(\rho_H) = 1$. Moreover, $D(\rho) > 1$ for $\rho \in (2s, \rho_H)$, whereas $D(\rho) < 1$ for $\rho \in (\rho_H, +\infty)$.

J Proof of Proposition 5

We note that substituting m^* given by (A.10) into (A.12), A_β can be rewritten:

$$A_\beta = \frac{s\epsilon_1 f(1)(\alpha\sigma/(1-\sigma) + 1 - (1-\tau)w)^{\frac{1}{\beta}}}{((1-\tau)w-1)^{\frac{1+\alpha}{\beta}}(\mu\tau w)^{\frac{1-\alpha}{\beta}}} \frac{\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}}{1 + \frac{\alpha}{(1-\tau)w-1}\frac{\theta\alpha\sigma/(1-\sigma)}{(1+\theta)(1+\theta+\alpha\sigma/(1-\sigma))}} \tag{A.21}$$

which means that A_β is decreasing in μ.

Consider first configuration 1 of the Proposition. Using the proof of Proposition 4, inequalities (A.18) are satisfied, with $A_\beta\beta$ arbitrarily close to 1. Since A_β is decreasing in μ, a slight increase of μ allows us to get $A_\beta\beta < 1$ and $C_\beta\beta + B_\beta < 1$, which rules out the occurrence of a Hopf bifurcation and implies that the steady state is stable for all $\rho \in (2s, +\infty)$.

Second, consider configuration 2 of the corollary. Inequalities (A.20) are satisfied, with $A_\beta\beta$ arbitrarily close to 1. Since A_β is decreasing in μ, a slight decrease of μ allows us to get $A_\beta\beta > 1$ and $C_\beta\beta + B_\beta > 1$, which rules out the occurrence of a Hopf bifurcation and implies that the steady state is unstable for all $\rho \in (2s, +\infty)$.

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